We Claim:

1. A method to assess whether a compound is an LDL clearance enhancing drug that includes mixing the drug with cholesterol-containing lipoprotein *in vivo* or *in vitro*; isolating the complex, and determining whether the binding of the compound to the complex causes a change in the three dimensional conformation of apoB-100 in the lipoprotein that enhances the binding affinity of the lipoprotein to the LDL receptor; wherein the LDL-clearance enhancing drug is not probucol or a mono- or di-ester of probucol, not a compound described in WO 98/09773, and not a silyl compound described in U.S. Patent Nos. 5,155,250 or 5,608,095.

2. The method of claim 1, wherein the cholesterol-containing lipoprotein is LDL.

3. The method of claim 1, wherein the cholesterol-containing lipoprotein is VLDL.

4. The method of claim 1, wherein the binding of the compound to the complex is determined by a sandwich ELISA.

5. The method of claim 1, wherein the binding of the compound to the complex is determined using agarose electrophoresis.

6. A method to alter the conformation of a cholesterol-containing lipoprotein comprising mixing the cholesterol-containing lipoprotein *in vivo* or *in vitro* with a compound and determining whether the binding of the compound to the complex causes a change in the three dimensional conformation of apoB-100 in the lipoprotein that enhances the binding affinity of the lipoprotein to an LDL receptor.

7. A method to determine whether a high plasma cholesterol level in a host is due to a genetic alteration of the host's apoB-100 protein comprising administering a LDL clearance enhancing drug to the patient, observing a lower than normal decrease in plasma cholesterol level, and then isolating and evaluating the host's apoB-100 protein.

8. A method to determine whether a high plasma cholesterol level in a host is due to a genetic alteration of the host's apoB-100 protein comprising exposing the host's apoB-100 protein to an LDL clearance enhancing drug in vitro under conditions in which the host's apoB-100 protein and the drug can form a complex, and then isolating and evaluating the change in conformation of the host's apoB-100 protein caused by any complexation.

9. A method to determine if a compound causes a change in the structure of apoB-100 in a cholesterol-containing lipoprotein that would be therapeutically useful, comprising carrying out a sandwich immunoreactivity assay in which an antibody directed to an epitope on

apoB-100 (known to be important to the LDL receptor binding process) as a capture antibody is laid onto a plate, the cholesterol-containing lipoprotein/test compound complex is added to the plate, and a second antibody, which can be polyclonal or monoclonal, is then used to quantify the amount of LDL complex captured.

10. A method to assess a conformational change in cholesterol-containing lipoprotein caused by complexation with a test compound comprising assessing the change in the electrophorectic mobility pattern of the cholesterol-containing lipoprotein using electrophoresis.

11. A method for lowering plasma cholesterol in a host comprising administering an effective amount of a compound that binds to cholesterol-carrying lipoprotein in a manner that alters the three dimensional configuration of the lipoprotein and increases the binding affinity of the apoB-100 protein to the LDL receptor; wherein the LDL-clearance enhancing drug is not probucol or a mono- or di-ester of probucol, not a compound described in WO 98/09773, and not a silyl compound described in U.S. Patent Nos. 5,155,250 or 5,608,095.

12. The method of claim 11, wherein the LDL receptor is on the surface of hepatic cells.

13. The method of claim 11, wherein the cholesterol-carrying liproprotein is LDL.

14. The method of claim 11, wherein the cholesterol-carrying liproprotein is VLDL.

15. A method for assessing whether a compound binds to a lipoprotein in a manner which lowers plasma cholesterol comprising complexing the compound with cholesterol-containing lipoprotein, isolating the resulting complex, and determining whether the binding of the compound to the complex causes a change in the three dimensional conformation of apoB-100 in the lipoprotein that enhances the binding affinity of the lipoprotein to the LDL receptor.

16. A method for lowering plasma cholesterol in a host comprising administering an effective amount of a compound that binds to cholesterol-carrying lipoprotein in a manner that alters the three dimensional configuration of the lipoprotein and increases the binding affinity of the apoB-100 protein to the LDL receptor in combination or alternation with a second drug that lowers cholesterol via a different biological pathway; wherein the LDL-clearance enhancing drug is not probucol or a mono- or di-ester of probucol, not a compound described in WO 98/09773, and not a silyl compound described in U.S. Patent Nos. 5,155,250 or 5,608,095.

17. The method of claim 16, wherein the LDL receptor is on the surface of hepatic cells.

18. The method of claim 16, wherein the cholesterol-carrying liproprotein is LDL.

19. The method of claim 16, wherein the cholesterol-carrying liproprotein is VLDL.

20. The method of claim 16, wherein the second drug is selected from the group consisting of a statin, a bile acid sequestrant, nicotinic acid, probucol, a fibrate derivative, Neomycin, and cholestyramine.

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